



Fig. 1. Skin biopsy showing pseudoepitheliomatous hyperplasia with inflammatory infiltrate, suggestive of halogenoderma (hematoxylin and eosin, $\times 145$).

tration of iodide-containing antitussive medications or contrast material, and on fluoroderma following fluoride ingestion in gels or toothpaste [4,5]. The histologic picture of halogen-induced damage is similar for all agents, consisting of pseudoepitheliomatous hyperplasia resembling well-differentiated squamous-cell carcinoma, but differing in its rapid development and the presence of inflammatory infiltrates [3]. The mechanism of tissue damage is unclear, but there are some indications of a hypersensitivity reaction [4]. Our patient had a necrotic skin lesion on his left forearm, developing soon after 2-CDA administration through an antecubital vein in close proximity to the lesion. 2-CDA contains chloride, and as the clinical and histologic appearance of the tissue closely resembles that of the halogenodermas, it is possible that the skin damage was caused by the chloride component of the drug. As the use of 2-CDA becomes more widespread, such rare adverse manifestations may be seen more often.

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**SHOSHANA ZEVIN
CHAIM HERSHKO**

*Department of Medicine, Shaare Zedek Medical Center,
Jerusalem, Israel*

ELIEZER ROSENMAN

*Department of Pathology, Hadassah Medical Center,
Jerusalem, Israel*

Shoshana Zevin, M.D., is now at the Division of Clinical Pharmacology and Experimental Therapeutics, San Francisco General Hospital, University of California, San Francisco, San Francisco, CA.

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Multiple Myeloma and AIDS

To the Editor: Patients with human immunodeficiency virus (HIV) infection can suffer from diverse forms of malignant hematological disease. However, the relationship between HIV infection and multiple myeloma is uncommon. We present 2 cases of patients with HIV infection and multiple myeloma.

Case 1, a 30-year-old male and former intravenous drug abuser, HIV group IV-C1 by CDC classification, was admitted to hospital due to progressive blindness in the right eye and general vesicular rash. Findings included Hb 7.7 g/dl (normal, 13-17 g/dl), VCM 101 fl (normal, 82-97 fl), white blood cell count (WBC) $3.21 \times 10^9/l$ (normal, $4-10.8 \times 10^9/l$), formula (83% segmented neutrophils, 10% lymphocytes, 2.5% monocytes, and 2.2% eosinophils), total CD4 $0.010 \times 10^9/l$, total CD8 $0.72 \times 10^9/l$, CD4/CD8 ratio 0.03, VSG 140 mm/hr, Ca 12.5 mg/dl (normal, 8.5-10.5 mg/dl), LDH 762 IU/l (normal, 150-450 IU/l), alkaline phosphate 1,377 IU/l (normal, 65-300 IU/l), total proteins 6.5 g/dl (normal, 6.5-8.7 g/dl), albumin 3.4 g/dl (normal, 3.5-5 g/dl), and gammaglobulin 18.6%, with normal electrophoresis. Lytic bone lesions in the ribs, shoulder blades, humerus, and both iliac crests were observed. Sternal bone-marrow aspiration showed myelodysplasia with megaloblastic signs, but a biopsy of the iliac crest gave evidence of an infiltration above 60% for atypical plasmatic cells. Immunohistochemical studies demonstrated a positive intracytoplasmatic lambda-chain clonality, which confirmed a diagnosis of myeloma.

Case 2, a 54-year-old male smoker (20 cigarettes per day), denied risk factors for HIV infection. He presented with anorexia and a progressive weight loss of 15 kg over the previous 2 months. Findings included Hb 5.9 g/dl (normal, 13-17 g/dl), VCM 75 fl (normal, 82-97 fl), reticulocytes $33.6 \times 10^9/l$ (normal, $25-70 \times 10^9/l$), WBC $12.9 \times 10^9/l$ (normal, $4-10.8 \times 10^9/l$), total proteins 6.2 mg/dl (normal, 6.5-8.7 mg/dl), albumin 2.5 mg/dl (normal, 3.5-5 mg/dl), and normal proteinogram. Anti-HIV and HIV-Ag were positive. Total CD4 was $0.320 \times 10^9/l$, total CD8 was $0.5 \times 10^9/l$, and CD4/CD ratio was 0.59. Skeletal X-ray survey showed lytic images in multiple locations. Analytical control showed an IgG kappa monoclonal gammopathy, IgG 2,250 mg/dl (normal, 639-1,349 mg/dl)/kappa chain 2,680 mg/dl (normal, 598-1,329 mg/dl), and concomitant Bence-Jones proteinuria. Bone-marrow aspiration gave evidence of atypical plasmatic cells, and a bone-marrow biopsy showed massive infiltration of the same cells as in the marrow aspirate. Monoclonality of tumoral cells was demonstrated by immunohistochemical study.

In recent years, isolated cases of multiple myeloma in patients with HIV infection [1-3] have been reported. The average age of patients with multiple myeloma and no HIV infection is higher than those infected with HIV. This could indicate that HIV infection plays a significant role in the evolution of this type of neoplasia. Various theories have been put forward in order to explain the etiopathogenesis of this association. The immunity of patients with HIV infection makes findings, such as polyclonal hypergammaglobulin and oligoclonal or mono bands common, but the distribution of subclasses of these chains is different from those that exist in myeloma. Konrad et al. [4] recently described the case of a patient with multiple myeloma and HIV infection, in whom paraprotein IgG-k directed against p24 antigen was detected. The viral theory has also been considered. Voelkerding et al. [5] found genetic sequences of EBV in the tumoral tissue of a patient with myeloma and AIDS. Multicenter studies that include a greater number

of patients with HIV infection will have to be carried out in order to establish the causes of this relationship.

XAVIER NOGUÉS
AUGUST SUPERVÍA
HERNANDO KNOBEL
JOSE LUIS LOPEZ-COLOMÉS

Department of Internal Medicine

SERGI SERRANO

Department of Pathology, Hospital del Mar, Barcelona, Spain

EUGENIA ABELLA

Laboratori de Referencia de Catalunya, L'Hospitalet, Barcelona, Spain

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